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Report

Entrainment of Prefrontal Beta Oscillations Induces an Endogenous Echo and Impairs Memory Formation

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Summary

Brain oscillations across all frequency bands play a key role for memory formation [1–4]. Specifically, desynchronization of local neuronal assemblies in the left inferior prefrontal cortex (PFC) in the beta frequency (~18 Hz) has been shown to be central for encoding of verbal memories [5–8]. However, it remains elusive whether prefrontal beta desynchronization is causally relevant for memory formation and whether these endogenous beta oscillations can be entrained by external stimulation. By using combined EEG-TMS (transcranial magnetic stimulation), we here address these fundamental questions in human participants performing a word-list learning task. Confirming our predictions, memory encoding was selectively impaired when the left inferior frontal gyrus (IFG) was driven at beta (18.7 Hz) compared to stimulation at other frequencies (6.8 Hz and 10.7 Hz) and to ineffective sham stimulation (18.7 Hz). Furthermore, a sustained oscillatory "echo" in the left IFG, which outlasted the stimulation period by approximately 1.5 s, was observed solely after beta stimulation. The strength of this beta echo was related to memory impairment on a between-subjects level. These results show endogenous oscillatory entrainment effects and behavioral impairment selectively in beta frequency for stimulation of the left IFG, demonstrating an intimate causal relationship between prefrontal beta desynchronization and memory formation.

Results

Brain oscillations in a wide range of frequencies play a core role in the formation of memories by coordinating neural activity in distributed cell assemblies [1–4]. Among these memory-related oscillations, a critical role seems to be played by desynchronized local neural activity in the beta frequency, which strongly correlates with episodic memory formation [5, 7, 9, 10]. A recent combined EEG-fMRI study [6] localized these beta power decreases (17–20 Hz) to the left inferior frontal gyrus (IFG), consistent with a plethora of fMRI studies implicating this region for memory formation [11, 12]. It has been argued that decreased beta oscillatory activity might help local cell assemblies to enhance information-coding capacity and thus aid memory formation [13]. However, no study has yet investigated whether a decrease in prefrontal beta oscillatory activity causally mediates memory formation. We therefore know little about the functional relevance of beta desynchronization for episodic memory. Applying rhythmic transcranial magnetic stimulation (TMS) to entrain neural oscillations [14], we here investigate this question in a simultaneous EEG-TMS experiment where participants performed a word-list learning task and received rhythmic TMS at different frequencies during encoding (Figure 1A).

Volunteers (n = 19; 10 females; age, 22.16; SD, 3.27) engaged in repeated study-test cycles (n = 16), wherein each cycle contained an encoding phase (20 words), a distracter phase, and a test phase (free recall). During encoding, participants were instructed to perform either an animacy task ("Is the word referring to a living or nonliving entity?") or an alphabet task ("Are the first and the last letters in alphabetical order?"). Trains of regularly spaced TMS pulses were delivered, starting 0.5 s after item onset (Figure 1A). Within each cycle, the frequency of rhythmic TMS was either 18.7 Hz, 10.7 Hz, or 6.8 Hz (counterbalanced). Importantly, the number of pulses (n = 18) was held constant between conditions. The frequency band of interest (18.7 Hz), stimulation site (left IFG, Figure 1B), and timing of stimulation were carefully chosen according to a previous combined EEG-fMRI study [6]. Control frequencies (10.7 Hz and 6.8 Hz) were selected to show minimal overlap in harmonics [15]. TMS was guided by neuronavigation via individual MRIs. An inactive sham condition at 18.7 Hz was conducted with the coil being tilted 90°, perpendicular to the scalp.

Driving the Left IFG at Beta Selectively Impairs Memory Formation

The effect of rhythmic TMS (18.7/10.7/6.8/sham) on performance of the encoding task (animacy/alphabet) was examined by means of accuracy rates. No significant main effect of TMS or interaction between TMS and TASK was observed (ps > 0.2). A significant main effect was obtained for TASK reflecting that participants made less errors in the animacy compared to the alphabet task (Figure 1C). Reaction time analysis indicated slowed reaction times for the 6.8 Hz condition, which probably is attributable to the fact that this stimulation lasted into the response time interval, thus artificially slowing reaction times (RT) (Figure S1A available online). Importantly, no significant difference in RT was observed between sham and 18.7 Hz stimulation (ps > 0.3).

Memory performance depending on TMS condition is shown in Figure 1D. A two-way ANOVA with the factors TASK (animacy/alphabet) and TMS (18.7/10.7/6.8/sham) revealed a significant main effect for TMS ($F_{3,54}$ = 3.32; p < 0.05). Post-hoc tests showed that this main effect was due to decreased memory performance in the 18.7 Hz condition compared to all other conditions (ts_{18} > 2.024; ps < 0.05). Therefore, rhythmic TMS at the left IFG impaired memory formation only when applied at beta. Intriguingly, memory performance in the other two active rhythmic TMS conditions was comparable to inactive sham stimulation (Figure 1D). A significant main effect for TASK was also obtained because of better memory performance in the animacy compared to the alphabet task ($F_{1,18}$ = 6.14; p < 0.05; see Figure S1B). No

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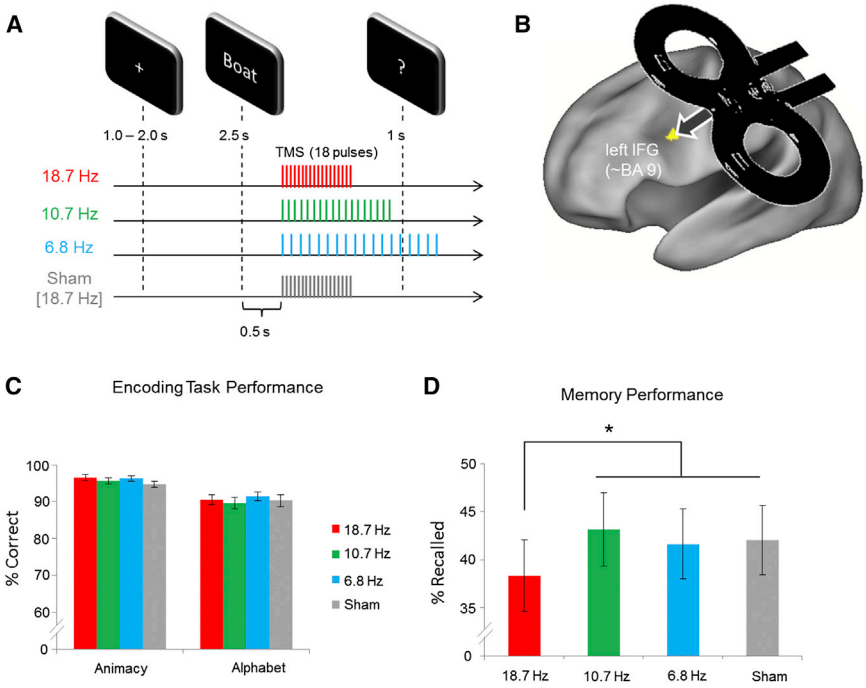

Figure 1. Task, TMS Conditions, and Behavioral Results

(A) The structure of a trial during encoding and the different rhythmic TMS conditions are shown. The question mark prompted the subjects to judge either whether the first and last letter of the presented word were in alphabetical order, or whether the presented word referred to a living or nonliving entity.

(B) The stimulated brain region, left IFG, MNI coordinates: x = −48, y = 9, z = 30 (peak voxel reported in Hanslmayr et al. [6] is shown).

(C) Performance during encoding for the two primary tasks (alphabet and animacy) is shown by means of accuracy rates.

(D) Behavioral results are shown for memory performance, split by TMS conditions. Note the drop in performance in the 18.7 Hz condition.

Error bars index mean SE.

significant TMS by TASK interaction was observed ($p > 0.5$). Together, these results demonstrate that rhythmic TMS at 18.7 Hz selectively impaired memory performance without affecting performance of the primary encoding task.

Driving the Left IFG at Beta Induces an Endogenous Oscillatory "Echo"

Oscillatory "echoes" were investigated by contrasting the EEG after rhythmic TMS offset between active and sham stimulation (Figure S2A). The assumption was that if rhythmic TMS indeed entrained an endogenous oscillation, thus forcing neural assemblies to resonate in the same phase, it should be visible in a phase-locked aftereffect as an entrainment echo (Figure 2A). Indeed, inspection of the band-pass filtered ERPs at left frontal electrode sensors exhibited such an echo at the behaviorally relevant beta frequency (Figure 2B) with a left frontal topography (Figure 2C). To investigate frequency specificity of this effect, ERP power [16] was examined. Smearing artifacts resulting from filtering were carefully controlled by excluding the first 400 ms after the last TMS pulse from analysis (see control analysis in Supplemental Experimental Procedures and Figures S2B and S2C). As a result of the high frequency of stimulation (18.7 Hz) and the strong artifacts induced by TMS, it was not feasible to analyze the EEG during stimulation itself (Figure S2A). Sham and real TMS conditions were contrasted in steps of 0.5 s for each frequency of interest (±1 Hz of the stimulation frequency), applying a nonparametric correction for multiple comparisons [17].

For the 18.7 Hz stimulation condition, a strong difference in ERP beta power (17.5–19.5 Hz) was evident between active TMS and sham in the first time window, 0.4 to 0.9 s after TMS offset ($p_{corr} < 0.005$). Consistent with the ERPs (Figures 2B and 2C) and with the stimulation site, the effect was maximal over left frontal sensors but was also visible, albeit to a weaker degree, at left parieto-occipital sites (Figure 3A). This 18.7 Hz echo was still significant from 0.9 to 1.4 s ($p_{corr} < 0.05$) and fell below significance in the last time window (1.4–1.9 s), showing that the echo gradually decreased over time as expected. This gradual decrease is also reflected in a polynomial contrast analysis indicating that beta ERP power, averaged over significant electrodes, across the three time windows followed a linear trend (mean percent signal change: 35.05, 25.26, and 18.24; $F_{1,18} = 6.902$; $p < 0.05$). Inspection of the frequency characteristics revealed that the beta TMS aftereffect was sharply centered at the stimulated frequency (Figure 3B). No such entrainment echoes were observed for the two other stimulation conditions, neither at 10.7 Hz nor at 6.8 Hz (Figures S2D, S3A, and S3B). The sources of the beta TMS echo were examined by means of a beamformer analysis [18], which was applied to the bandpass filtered ERP data. Source analysis suggests that the beta TMS echo was confined to neural assemblies in the left IFG including the stimulated region (Figure 4A). Thus, the frontal-occipital topography probably was generated by dipoles in this region. Together, these data demonstrate the existence of an endogenous beta oscillatory echo in response to 18.7 Hz stimulation, outlasting the stimulation period by approximately 1.5 s, and suggest that local neural assemblies in the left IFG during memory formation preferably entrain to a beta rhythm but not to other rhythmic stimulation.

Specificity of Beta Echoes and Relation to Behavior and to Endogenous Beta Frequency

To investigate whether the left IFG always responds with a sustained 18.7 Hz echo when stimulated with TMS, irrespective of stimulation frequency, we investigated to which extent beta ERP aftereffects (17.5–19.5 Hz; 0.4–0.9 s) were also evident in the two other active TMS conditions. Although a trend for enhanced beta activity after active TMS compared to sham stimulation was visible, no significant aftereffect emerged in any of the two conditions (p_{corr}s > 0.05, Figure S3C). Importantly, directly contrasting the TMS aftereffects between the 18.7 Hz condition and the other two active TMS conditions on the source level ($p_{corr} < 0.005$; Figure 4B) and at the sensor level ($ts_{18} > 1.92$; $p < 0.05$; Figure 4C) revealed stronger beta entrainment for the 18.7 Hz condition compared to the 10.7 and 6.8 Hz condition (see also Figures S3D and

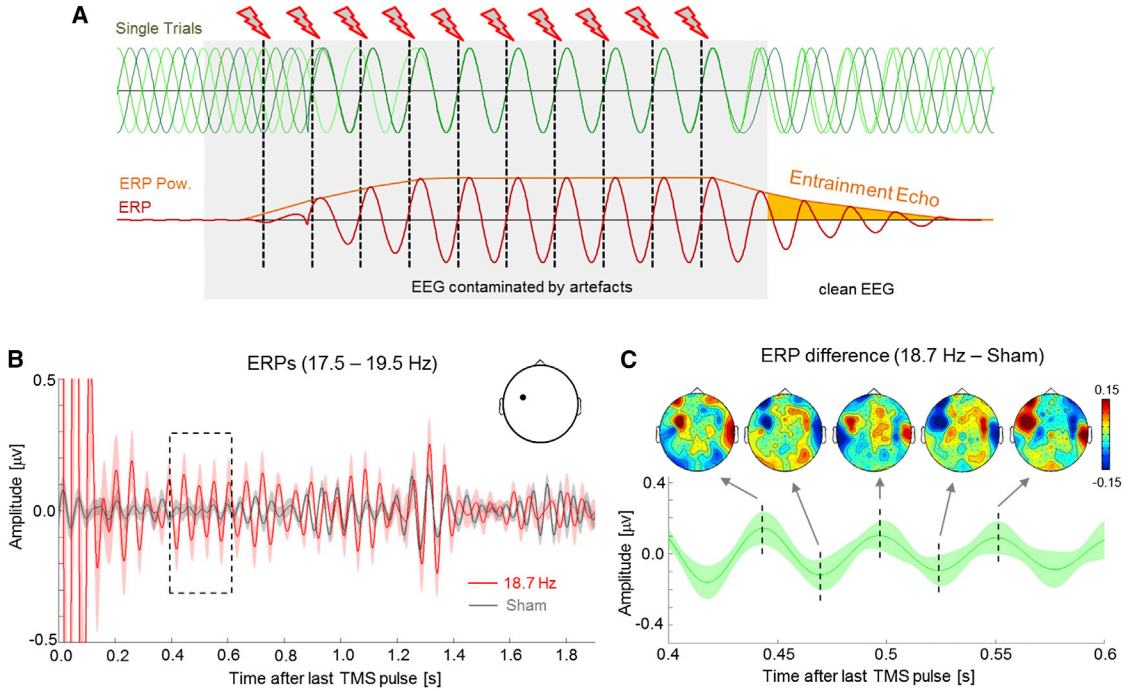

Figure 2. EEG Entrainment Echoes and ERPs

(A) A schematic of the hypothesized effect of TMS entrainment is shown. EEG single trials (top panel) are out of phase prior to TMS stimulation and gradually align in phase during stimulation. Phase consistency between trials outlasts the stimulation (echo) before slowly decaying to baseline. This entrainment echo is visible in the ERP, and in ERP power at the entrained frequency (lower panel).

(B) ERPs (grand average) for the 18.7 Hz and sham conditions are shown for a representative left frontal EEG channel. ERPs are band-pass filtered from 17.5 to 19.5 Hz to visualize the echo effect. Shaded areas indicate mean SE.

(C) Box area in (B) indicates the time window for which the difference between the 18.7 and sham ERPs is shown. Note that the maximal difference occurs at left frontal electrodes.

S3E). Notably, these results further argue against a nonphysiological source of the beta echoes, e.g., filter smearing of the TMS artifacts that are present in all three active TMS conditions.

To evaluate whether the beta entrainment echoes were related to TMS-induced memory impairment, participants were split based on their differences in evoked beta power (18.7 Hz versus sham). Presumably in some subjects entrainment might have been more effective than in others, which should be evident in the entrainment echoes. If beta entrainment bears a functional relationship to memory formation, high-entraining subjects should show stronger memory impairment than low-entraining subjects. This analysis was performed separately for the two time windows where significant beta echoes emerged (0.4–0.9 and 0.9–1.4 s). Indeed, stronger TMS-induced memory impairment for high compared to low entrainers was obtained ($t_{18} = 1.81$; $p < 0.05$; Figure 4D). However, this effect was present only in the later (0.9–1.4 s), not in the earlier (0.4–0.9 s), time window ($p > 0.3$). These results corroborate a functional role for beta entrainment echoes and suggest that participants with stronger and longer-lasting echoes show more memory impairment than participants with only weak and short-lived entrainment echoes.

Finally, we investigated whether endogenous oscillatory power in the stimulated beta frequency (~18.5 Hz) predicted memory formation and whether it interacted with the effects of beta TMS. Accordingly, a subsequent memory analysis was performed contrasting EEG power for items that were later remembered with items which were later not remembered [12]. Analysis was restricted to the sham stimulation condition and to the time-window prior to TMS onset (0–0.5 s, see Supplemental Information and Figure S4A). Replicating previous findings [6], a stronger beta power decrease was obtained for later-remembered compared to later-forgotten items (Figure S4B). The peak frequency of this effect across subjects was 18.5 Hz (SD 1.7 Hz), in line with the observed echo frequency (Figure S4C). Furthermore, subjects with a low deviation (≤1 Hz) in this beta peak frequency from the stimulated frequency exhibited stronger and longer-lasting beta echoes than did subjects with a high deviation (>1 Hz) in peak frequency (Figure S4D). Together, this analysis shows the existence of a memory-relevant internal oscillation at the stimulated frequency that strongly interacts with the effects of entrainment.

Discussion

The current study demonstrates that artificially synchronizing the left IFG in the beta frequency range interferes with episodic memory encoding. Memory was impaired only when the left IFG was synchronized in the beta frequency range, and not at other frequencies. Crucially, task performance at encoding was unaffected by beta TMS. An oscillatory echo after stimulation was evident only after the behaviorally relevant beta frequency TMS, which was related to memory impairment. Intriguingly, memory performance in the two other active TMS conditions was indistinguishable from inactive sham stimulation. This pattern of results is remarkable because it demonstrates that even when a highly-task-relevant region is actively stimulated with TMS, behavioral effects are observed

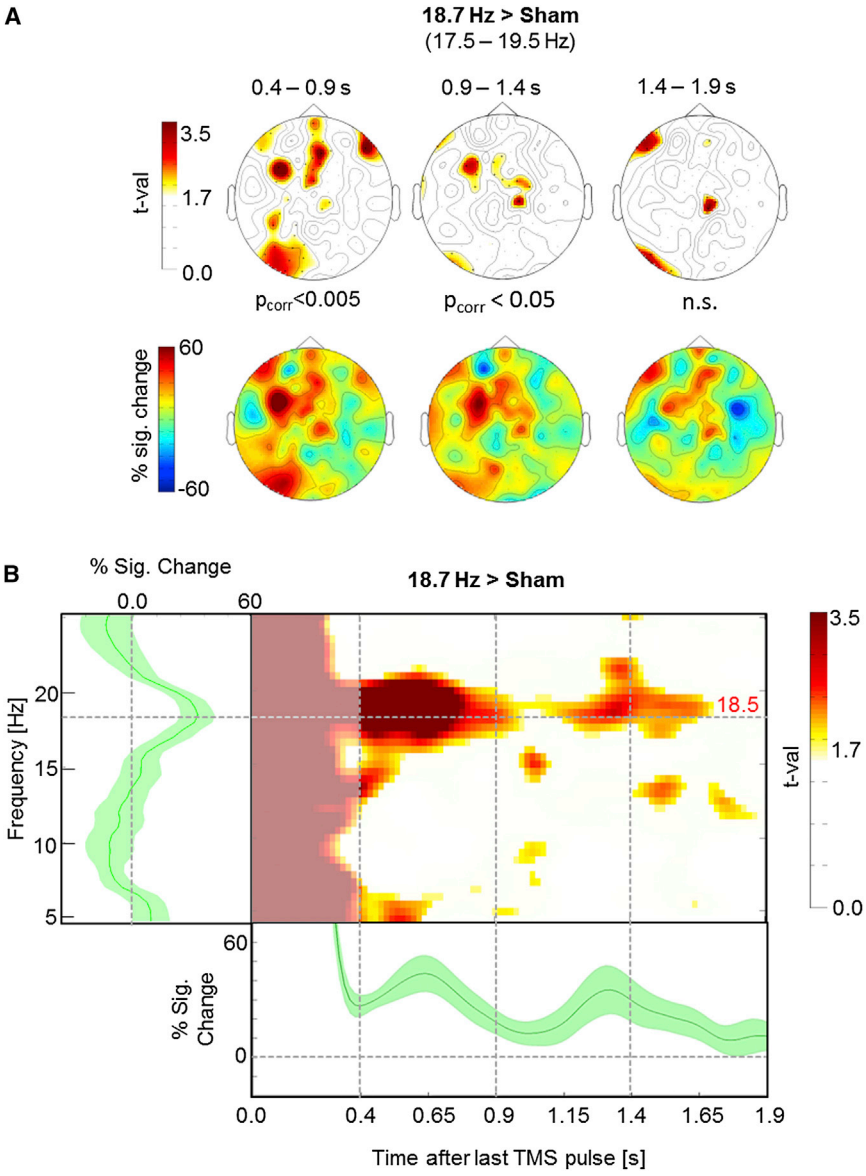

Figure 3. ERP Power

(A) The topographical distribution of ERP power difference between 18.7 Hz and sham stimulation is shown in steps of consecutive 0.5 s time windows for three time intervals after the last TMS pulse (discounting the segment containing filter artifacts). Differences are shown in terms of statistical maps (t-values) and absolute differences in ERP power (% signal change). Note the left frontal topography of the effect.

(B) A time-frequency plot of the differences in ERP power (18.7 Hz > sham) averaged over the significant sensors (0.4–0.9 s) is shown. The transparent area (0–0.4 s) marks the period containing the TMS artifact smeared by the filter (see Figure S2B). The adjacent plots show differences in ERP power between 18.7 Hz and sham condition (shaded areas reflect SE). The entrainment echo is sharply centered at the stimulation frequency and slowly decays over time.

only when TMS is optimally tuned to the frequency characteristics of that region in that particular task [19].

How can the frequency-specific effects of TMS stimulation on behavior be explained on a mechanistic level? Our EEG results show that the left IFG has a preferred resonant frequency in the beta range. Enhanced beta oscillations typically index inactivity or rest [20] or pathological conditions [21]. Recent studies in monkeys demonstrate that neurons in the prefrontal cortex dynamically synchronize and desynchronize at beta during executive functions [22] and that the spiking of prefrontal neurons relative to beta phase codes items in working memory [23]. Enslaving large prefrontal neural assemblies to the same stereotypical beta rhythm via rhythmic TMS probably prevents these locally fine-tuned beta dynamics, disabling the processes required for memory formation. Arguably, stimulation at the two other task-irrelevant frequencies did not interfere with these task-relevant beta dynamics and therefore did not affect encoding. Notably, three previous TMS studies observed memory impairment when stimulating the left PFC at 5 Hz and 10 Hz [24–26]. These studies, however, differed in several respects from the current one (e.g., timing, task) and targeted quite different regions in the left PFC (more dorsal and anterior) than in the current study. On a conceptual level, our results corroborate the recently proposed Information via Desynchronization Hypothesis, suggesting that memory-related decreases in oscillatory activity enhance the system's ability to code information in the episodic memory system [13].

A previous rhythmic TMS study found entrainment of brain oscillations only online to TMS; they did not observe entrainment echoes [14]. When reporting such oscillatory echoes, it is important to show (1) that these TMS aftereffects are not due to filter smearing (see Figures S2B and S2C and Supplemental Discussion), (2) that they outlast the TMS-evoked potential elicited by the last pulse, and (3) that they depend on the frequency of stimulation. We controlled for these aspects by discarding the first 400 ms after TMS offset (TMS-evoked potentials typically do not last longer than 400 ms [27]) and by showing that the beta entrainment echo was stronger after beta stimulation compared to the other active TMS conditions (Figure 4B; see also Figures S3B and S3C). Our result of a sustained prefrontal beta entrainment echo (approximately 1.5 s) thus strongly supports the idea that endogenous oscillations can be entrained with external rhythmic stimulation [19]. A likely reason for why a previous study [14] observed entrainment only during rhythmic TMS and no entrainment echoes is that they used only 5 TMS pulses, whereas 18 pulses were used in the current study.

When testing for frequency-specific effects of rhythmic TMS, one can control for either the duration of stimulation or the number of pulses. To control for the amount of energy that is delivered to the IFG, we decided to match the number of pulses across stimulation conditions. However, this inevitably introduces a possible confound between stimulation

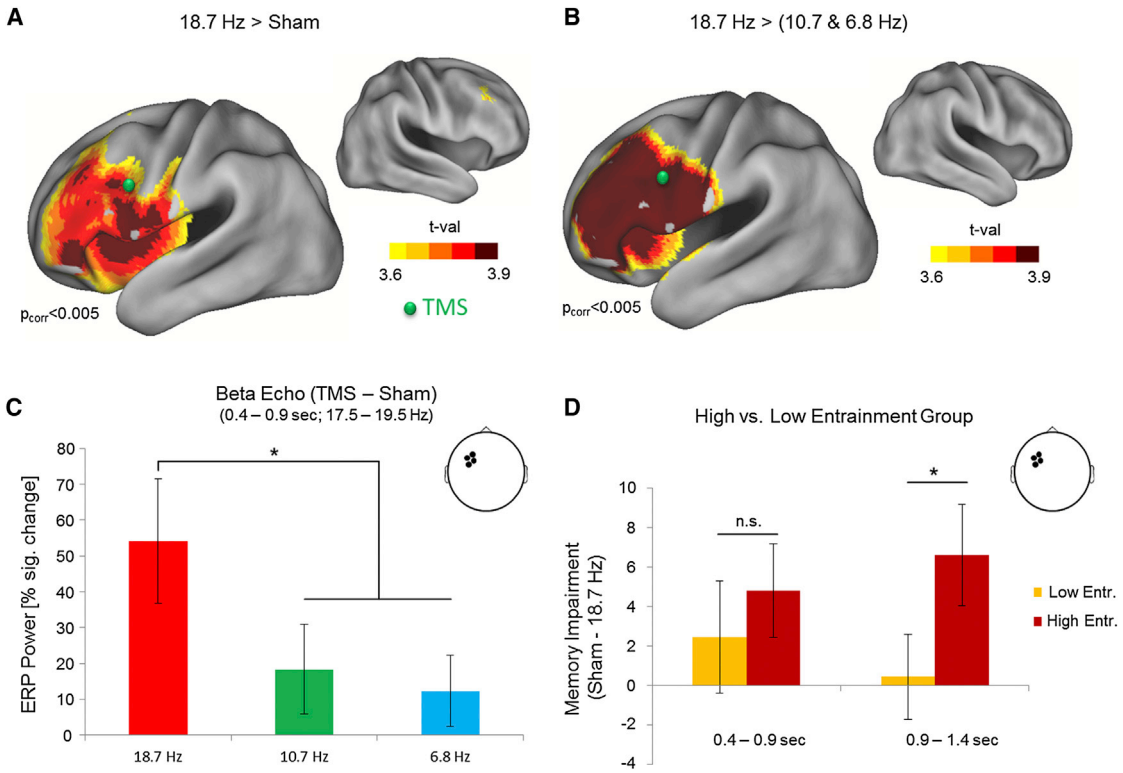

Figure 4. Sources of Entrainment Echoes and Relation to Behavior

(A) Source localization results of the beta ERP power (17.5–19.5 Hz, 0.4–0.9 s) contrasting the 18.7 Hz condition with sham stimulation is shown. The entrainment echo is confined to left inferior prefrontal regions.

(B) The difference in ERP power (17.5–19.5 Hz; 0.4–0.9 s) between the 18.7 Hz condition versus the other two active rhythmic TMS conditions (10.7 Hz and 6.8 Hz) is shown. The green spheres highlight the site of TMS.

(C) Beta oscillatory echoes, i.e., differences in ERP power (17.5 to 19.5 Hz) between TMS and sham, are shown for each of the three active stimulation conditions. ERP power values are averaged across a selected group of left frontal EEG sensors.

(D) The difference between high- and low-entraining subjects in rhythmic TMS-induced memory performance is shown. Participants were split based on the beta entrainment echoes (18.7 Hz versus sham) using ERP power in the two significant time windows of the four most significant left prefrontal EEG sensors (upper right).

Error bars index mean SE.

frequency and stimulation duration, which needs to be considered because the timing of TMS strongly impacts on behavior [28]. Nevertheless, the condition with the shortest stimulation demonstrated the strongest impairment of memory. Another possible confound is discomfort introduced by rhythmic TMS resulting from peripheral muscle stimulation (e.g., faster frequencies might be more painful than slower stimulations). However, subjective ratings of discomfort in our data set indicate similar pain levels across the three stimulation frequencies ($p > 0.5$; Figure S1C). Taken together, it is thus very unlikely that the observed effects can be explained by unspecific TMS effects.

There are several open issues that deserve further attention in future studies. First, no stimulation was applied at frequencies faster than beta because of ethical [29] and technical limitations of TMS. We can therefore not rule out that stimulation at higher frequencies might have been even more detrimental. Future studies using transcranial alternating current stimulation (tACS) [30, 31] could explore this question. Second, the time windows during stimulus processing that were used for investigating oscillatory echoes differed between the three active TMS conditions (6.8, 10.7, and 18.7 Hz) because of the different durations of stimulation (see Supplemental Discussion). Future studies could employ an arrhythmic stimulation condition, where trains of pulses are delivered jittered in time but with equal durations, to circumvent this problem. Third, although previous studies suggest that encoding of verbal material is impaired only when the left IFG, but not when the right IFG, is stimulated with beta TMS [32], future studies should also employ the echo effects when different brain regions are driven at beta.

Taken together, the current study provides behavioral and electrophysiological evidence for a frequency-specific effect of prefrontal rhythmic TMS on memory formation. Specifically, our results demonstrate that stimulation of the prefrontal cortex at the task-relevant beta frequency interfered with memory encoding and induced a sustained oscillatory echo. These data suggest a new way of measuring entrainment effects in the human brain—oscillatory echoes. Most importantly, our results demonstrate, for the first time, that prefrontal beta desynchronization is more than an epiphenomenon and plays a causal role for memory formation.

Supplemental Information

Supplemental Information includes Supplemental Discussion, Supplemental Experimental Procedures, and four figures and can be found with this article online at http://dx.doi.org/10.1016/j.cub.2014.03.007.

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